PERSONAL PRACTICE

Supraventricular tachycardia: diagnosis and current acute management

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There is a small but potentially avoidable incidence of death associated with childhood supraventricular tachycardia.[1 2] The time of greatest risk is when the child presents for the first time and requires therapeutic intervention. Infants and children tolerate arrhythmias less well than adults as they are more dependent on heart rate for cardiac output and have less reserve.[3 4] They are also at risk from drugs given as treatment.[5] We discuss our current acute management of supraventricular tachycardia.

Diagnosis

Between 30 and 40% of children who present with supraventricular tachycardia do so within the first few weeks of life. Their presentation is variable. Supraventricular tachycardia can be the cause of unexplained hydrops of the fetus or can result in sudden profound cardiovascular collapse in the newborn period.[6] More usually neonates and small infants will present with symptoms of increasing tachypnoea, poor feeding, and pallor which have developed over a few days. Occasionally supraventricular tachycardia is intermittent and a strong index of suspicion must be maintained if the diagnosis is not to be missed. The older child more usually presents with a history of palpitations.

The most common type of supraventricular tachycardia in childhood results from a re-entry circuit between atria and ventricles involving the atrioventricular node as the antegrade limb and an accessory connection between ventricle and atrium as the retrograde limb (fig 1). This type of tachycardia, atrioventricular re-entry tachycardia, can be classified as a junctional supraventricular tachycardia (table). An accessory connection may occur anywhere around the atrioventricular ring. If it is capable of supporting conduction antegradely from atrium to ventricle, as well as retrogradely, the usual situation during supraventricular tachycardia, it will be manifest on the electrocardiogram during sinus rhythm as a short PR interval and delta wave. Such pre-excitation of the ventricle together with supraventricular tachycardia is the syndrome described by Wolff, Parkinson, and White.[7] If conduction is restricted to the retrograde direction, atrioventricular re-entry tachycardia is possible but there will be no evidence of the connection on the electrocardiogram during sinus rhythm, a situation termed concealed pre-excitation.

The majority of children with an accessory

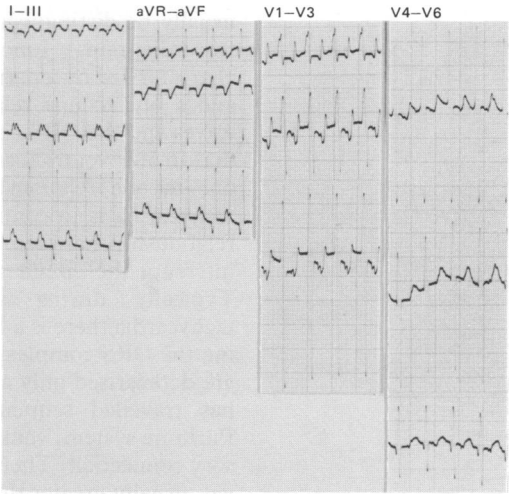

Figure 1 An electrocardiogram recorded from a 1 week old neonate with atrioventricular re-entry tachycardia showing the typical pattern with a P' wave almost midway between QRS complexes.

Classification of supraventricular tachycardia

Junctional tachycardias

Atrioventricular re-entry tachycardia (AVRT)

Orthodromic AVRT:
Tachycardia involving a circuit utilising the atrioventricular node as the antegrade limb and an accessory connection between ventricle and atrium as the retrograde limb

Antidromic AVRT:
Tachycardia involving a circuit utilising an accessory connection as the antegrade limb and the atrioventricular node as the retrograde limb

Atrioventricular nodal re-entry tachycardia (AVNRT)

Tachycardia involving a circuit the two limbs of which are intimately associated with the atrioventricular node. The atria and ventricles are activated as offshoots of the circuit. The common type of AVNRT is typified by slow conduction antegradely and fast conduction retrogradely, the uncommon type by fast conduction antegradely and slow conduction retrogradely

Long R–P' tachycardia

Tachycardia involving a circuit utilising the atrioventricular node as the antegrade limb and a slow conducting pathway as the retrograde limb. The latter may be an accessory connection in close association with the atrioventricular node, in the posteroseptal position or remote from the atrioventricular node

Atrial tachycardias

Atrial tachycardia

Tachycardia characterised by discrete atrial activity on the surface electrocardiogram with varying ventricular response resulting either from a micro re-entry circuit or ectopic focus confined within the atria

Atrial flutter

Tachycardia characterised by a sawtooth undulation of the baseline on the surface electrocardiogram probably resulting from conduction around a re-entry circuit within the atria

Atrial fibrillation

Tachycardia characterised by chaotic low voltage 'fibrillatory' waves on the surface electrocardiogram with an irregularly irregular ventricular response, resulting from disordered atrial actvity

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connection do not have structural congenital heart disease. When there is a structural anomaly this is most commonly Ebstein's anomaly of the tricuspid valve or less frequently, corrected transposition (discordant atrioventricular connection, discordant ventriculoarterial connection).

Atrioventricular re-entry tachycardia accounts for the great majority of supraventricular tachycardias presenting in the first year of life. The spectrum of supraventricular tachycardia in older children is wider and both atrial and other junctional tachycardias such as atrioventricular nodal re-entry tachycardia or long R-P' tachycardia are more frequently seen. They can usually be distinguished from the surface electrocardiogram. Junctional tachycardias are characterised by a narrow regular QRS complex and a one to one relationship between the P' waves and the QRS complexes during tachycardia. In atrioventricular re-entry tachycardia this one to one relationship is a necessity if the tachycardia is to continue, as disruption of the relationship between atrium and ventricle will interrupt the circuit and stop the tachycardia. Typically during atrioventricular re-entry tachycardia there is a P' wave easily seen following the QRS complex. This is because the atria are depolarised only after the electrical impulse has travelled sequentially through the His-Purkinje system, ventricular muscle, and accessory connection. There is therefore a time interval, usually greater than 70 msec, between ventricular and atrial depolarisation. In contrast, during atrioventricular nodal re-entry tachycardia the atria and ventricles are both activated as offshoots of a re-entry circuit involving a fast and slow pathway in close association with the atrioventricular node. The retrograde pathway is typically fast and therefore atria and ventricles are depolarised almost simultaneously. As a result the P' wave is not easily seen, being hidden from view by the QRS complex. The other less common junctional tachycardia, the long R-P' tachycardia, can often be distinguished by its 'persistent' nature, stopping and starting during the electrocardiogram recording, and the characteristic superior P' wave axis, seen just before the QRS complex (fig 2). This electrocardiographic pattern represents a re-entry circuit involving the atrioventricular node antegradely and a slow conducting pathway, which may either be an accessory pathway or part of the atrioventricular node, retrogradely. It is this long interval between the R wave of the QRS complex and the P' wave, representing slow conduction in the retrograde pathway, that gives the tachycardia its name.[8]

Atrial arrhythmias, for example, atrial tachycardia, atrial fibrillation, or flutter, are not often seen in the first year of life. They are more common, especially after cardiac surgery, in the older child with structural heart disease where the atria are distorted or distended, or in the child with myocarditis. They may occasionally be seen in association with an accessory connection. Unlike the more common junctional tachycardias they can continue in the presence of atrioventricular nodal block; a one to one relationship of atrial to ventricular depolarisation is not required.

Before any treatment is administered the tachycardia should be documented by recording a 12 lead electrocardiogram and rhythm strip. This is important as correct diagnosis of the arrhythmia should guide treatment and prevent use of inappropriate or potentially hazardous drugs. Ventricular tachycardia should be excluded. Ventricular arrhythmias are relatively uncommon in childhood. When they do occur

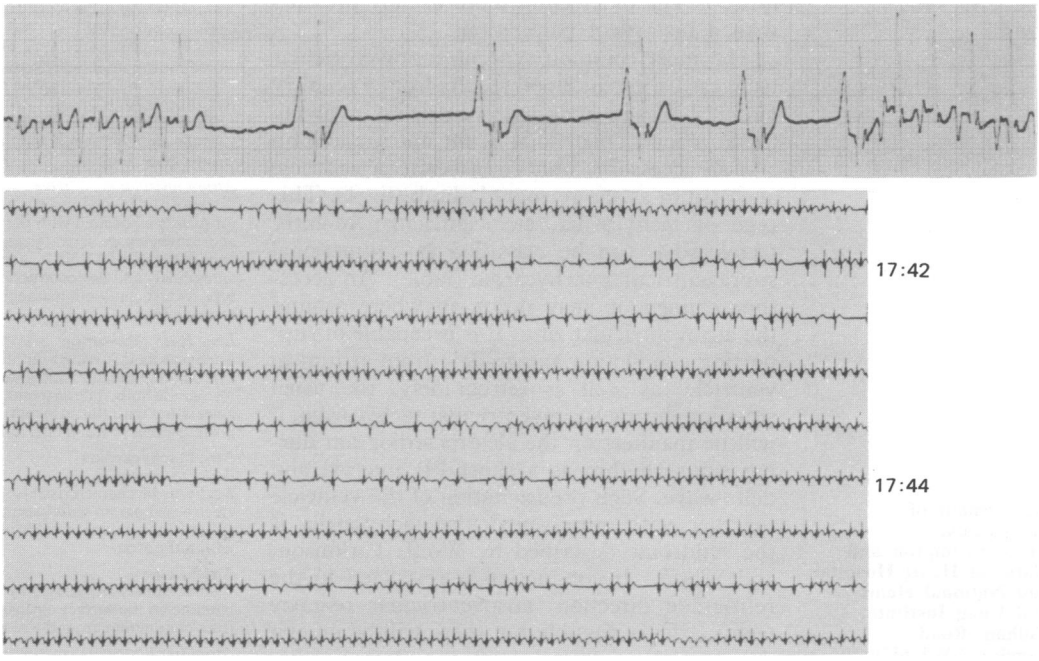

Figure 2 A section from a 24 hour tape recording performed on an infant with a long R–P' tachycardia. The electrocardiogram (top) (25 mm/sec), shows the tachycardia with P' wave (inverted) just before the QRS complex, terminating spontaneously. There follows four beats of sinus rhythm before the tachycardia reinitiates. The three minute sequence shown below shows how this behaviour continues repeatedly.

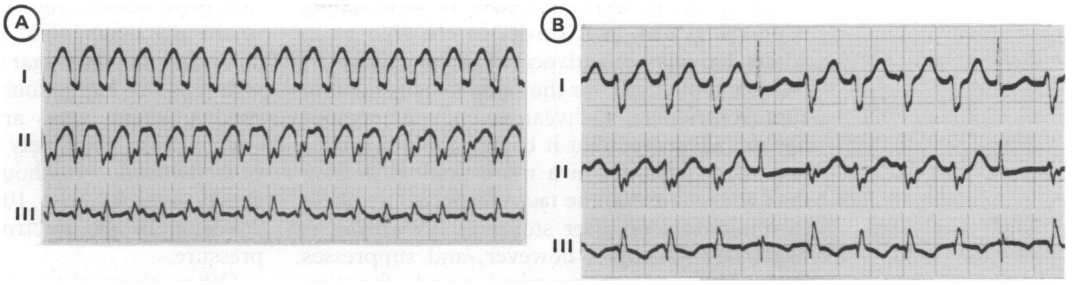

Figure 3 (A) Leads I, II, and III from an electrocardiogram recorded from a 1 year old child with ventricular tachycardia. The child had an otherwise structurally normal heart, no electrolyte inbalance, and was haemodynamically stable at the time of the recording. The rate is 320 bpm and atrioventricular dissociation can be seen. (B) Similar leads taken from an electrocardiogram recorded from the same child some hours later after a spontaneous slowing of ventricular tachycardia rate. Capture beats are now seen (4th and 9th QRS complex).

they are most frequently only recognised after a cardiac arrest as a result of inappropriate drug treatment given in the mistaken belief that the arrhythmia being treated was supraventricular tachycardia.[5] Unfortunately the electrocardiographic appearances of ventricular tachycardia are not typical in early life. The QRS complex is not neccessarily grossly widened and the rate may exceed 300 beats/minute (bpm). Helpful signs are an abnormal QRS axis for age, the presence of fusion or capture beats and atrioventricular dissociation (fig 3). It is rare for supraventricular tachycardia to have a wide QRS complex in childhood but may be seen in association with Ebstein's anomaly,[2] after cardiac surgery requiring a ventriculotomy or after treatment with antiarrhythmic drugs. Then the morphology of the QRS complex closely resembles that of either right or left bundle branch block (more commonly right). Comparison with previous electrocardiograms, if available, is invaluable as the presence of bundle branch block most often predates the onset of tachycardia. It is comparatively rare to develop bundle branch block as a result of tachycardia.

Treatment

The ideal acute management of supraventricular tachycardia is a subject hotly debated by cardiologists, but in practice, one that depends on facilities locally available. If the child is tolerating the arrhythmia well and the supraventricular tachycardia is judged to be one of the more common junctional tachycardias, atrioventricular re-entry tachycardia or atrioventricular nodal re-entry tachycardia, there are a number of options available, any of which are acceptable. Our current recommendations are outlined in fig 4.

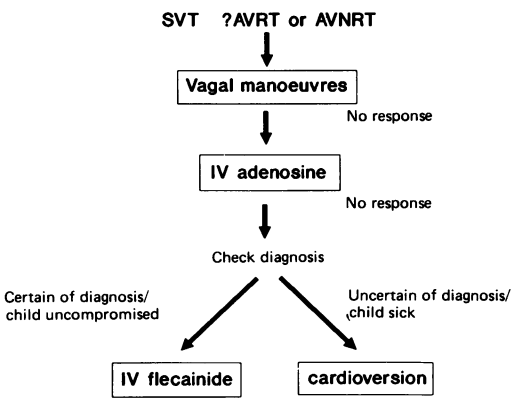

Figure 4 Acute management of supraventricular tachycardia (SVT) in childhood: current recommendations. AVRT, atrioventricular re-entry tachycardia; AVNRT, atrioventricular nodal re-entry tachycardia.

Vagal manoeuvres are easy to perform, quick, safe, and often successful.[9] Immersion of an infant's face in cold water, to elicit the diving reflex, appears to be very effective.[10] Some older children will actively participate in vagal manoeuvres, particularly if it is not their first attack. Others are too frightened and attempts at immersing their face in cold water do little to improve this. If vagal manoeuvres fail drug treatment may be considered.

Digoxin has been used to treat supraventricular tachycardia for many years. It has advantages in that it is familiar and is not negatively inotropic. More recently digoxin has fallen from favour because of the danger of inappropriate treatment of ventricular tachycardia, where it may precipitate ventricular fibrillation, and because of the slowness of its action (median time six hours).[11] In preference therefore we would chose adenosine as the drug of first choice for the treatment of acute supraventricular tachycardia. Adenosine is a new drug with many attractive characteristics. It acts by slowing atrioventricular nodal conduction thus disrupting a re-entry circuit (fig 5). It has a rapid onset of action and is effective within 10 to 20 seconds of being given intravenously in approximately 86% of junctional tachycardias.[12–14] It has a short half life of 10–15 seconds so side effects, which occur in one third of patients treated, are transient and rarely require intervention. Additionally adenosine is not negatively inotropic in this form and so may be given to an infant or child in low cardiac output without fear of exacerbating this. Moreover if adenosine is administered to a child with ventricular tachycardia by mistake it will not precipitate ventricular fibrillation. Indeed a bolus of adenosine can be used in the difficult situation of a wide complex tachycardia as a diagnostic aid to help distinguish ventricular from supraventricular tachycardia.[15] The main disadvantage of using adenosine is that in approximately 30% of cases the tachycardia will reinitiate. None the less because of its high safety profile it is our drug of first choice.

Verapamil is highly effective in terminating supraventricular tachycardia in children as in adults and has been advocated in the past as the treatment of choice for the acute management of supraventricular tachycardia.[16–18] Verapamil has the advantage that it brings about termination of tachycardia in a more gradual fashion than adenosine and the tachycardia is less likely to reinitiate soon after stopping. Verapamil is negatively inotropic, however, and suppresses both sinus and atrioventricular node function and within the last few years reports have been published of infants reacting to verapamil with profound bradycardia and hypotension.[19–21] In some cases this has been irreversible despite the use of calcium. Deaths such as these have caused physicians to suggest that verapamil should never be used in infancy. Undoubtedly some of the reported cases have been due to inappropriate use, such as following the administration of β blockers or in infants with known severe ventricular dysfunction,[5] but there is some evidence to support the notion that young infants are more sensitive to verapamil than adults.[22] As there appears to be a safe alternative in the form of adenosine we have adopted the latter drug when treating infants less than 1 year of age.

Flecainide is a further antiarrhythmic drug which may soon be licensed for use in children in this country. Like other class 1c drugs it has a different mode of action from the drugs so far discussed, exerting profound effects on the accessory connection as well as the atrioventricular node. Typically when used to terminate supraventricular tachycardia, the arrhythmia will be seen to terminate with a QRS complex as retrograde conduction in the accessory connection is prevented (fig 5). It is useful, therefore, in the management of those tachycardias that are refractory to vagal manoeuvres or adenosine.[23] It also has actions on the atria and can be used to treat atrial arrhythmias.[24] However flecainide is negatively inotropic and can be proarrhythmic. It should therefore be given slowly over at least 10 minutes with careful attention to the electrocardiogram and blood pressure.

Other therapeutic options available when dealing with the acute situation are direct current (DC) cardioversion or pacing, either via an oesophageal or transvenous electrode. If the child is sick and a rapid response with no further depressant effect on cardiac output is required vagal manoeuvres can still be tried. Intravenous adenosine is safe and effective, if readily available (see footnote), but if not, DC cardioversion should be tried. We use one joule/kg in the first instance. If the QRS complex is wide but supraventricular tachycardia is suspected, vagal manoeuvres or intravenous adenosine can still be safely given. Because of the risk that a wide QRS complex tachycardia may be ventricular in origin verapamil and digoxin should *not* be used. Pacing techniques can be very effective and are relatively easy to apply,[25 26] but equipment is usually available only in cardiac centres.

Of the many other antiarrhythmic drug available all have potential disadvantages. In the acute situation when vagal manoeuvres and adenosine have failed we use flecainide if ventricular function is not seriously impaired. If ventricular function is impaired we would try DC cardioversion or pacing to prevent further deterioration. We chose flecainide because it is a

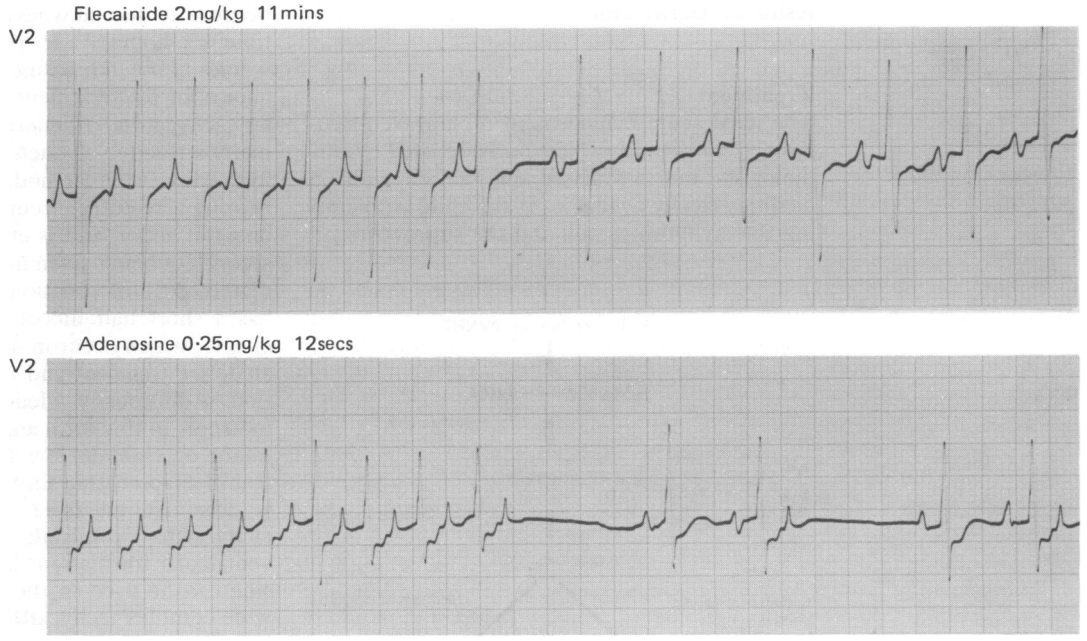

Figure 5 Two rhythm strips of lead V2 recorded from a child with atrioventricular re-entry tachycardia. The upper strip is recorded after the administration of intravenous flecainide showing the typical response of a slowing tachycardia rate followed by termination. Termination characteristically occurs in the limb of the circuit formed by the accessory connection (the tachycardia stops after a QRS complex). The lower strip is a recording from the same child on a different occasion after the administration of intravenous adenosine. The tachycardia terminates in the atrioventricular nodal limb of the circuit (the tachycardia stops after a P' wave), a sinus beat follows, and then the next sinus beat is followed by an atrial echo beat. This pattern continues until sinus rhythm resumes.

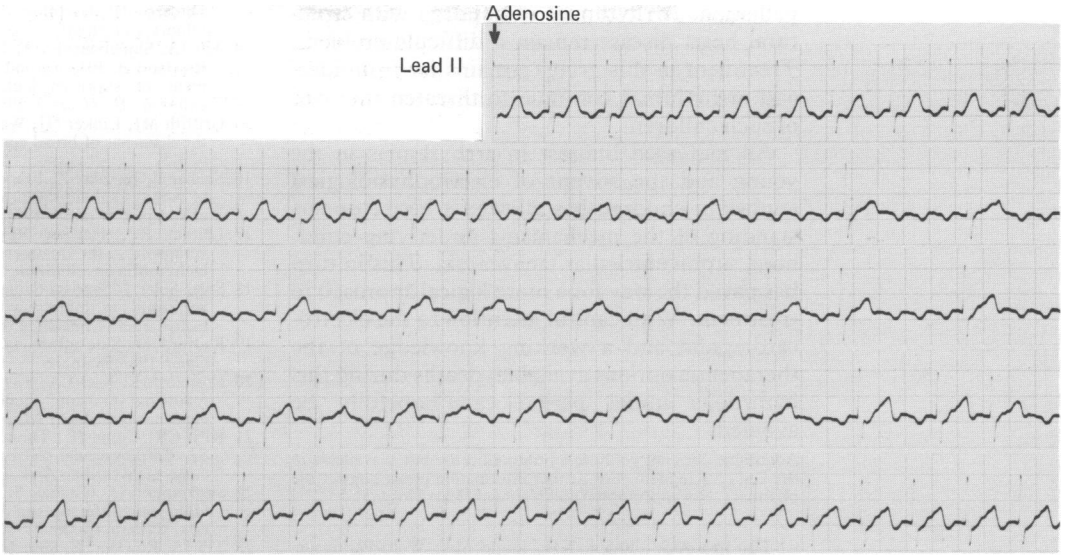

Figure 6 Electrocardiogram recorded from a child with atrial flutter showing the typical response of atrial flutter to intravenous adenosine. Adenosine fails to stabilise the atria but causes a transient slowing of ventricular rate as a result of an increased degree of atrioventricular nodal block, allowing confirmation of the diagnosis.

class 1 drug with with which we are familiar. Preliminary studies in children using propafenone are also favourable.

Atrial arrhythmias, in contrast to junctional, seldom respond to vagal manoeuvres and atrioventricular nodal blocking drugs such as adenosine or digoxin merely slow the ventricular rate rather than stopping the tachycardia (fig 6). DC cardioversion may be more effective at restoring sinus rhythm in this situation. Otherwise a class 1 drug such as flecainide may be necessary to stabilise the atria. If digoxin is used in an attempt to slow the ventricular rate, underlying Wolff-Parkinson-White syndrome must be excluded first, as digoxin may allow an increase in the conduction of atrial impulses to the ventricle via the accessory connection thus precipitating ventricular flutter or fibrillation.[27 28]

For those children with structural congenital heart disease particularly those who develop arrhythmias late after surgery management is less easy. Surgery may leave a child with ventricular dysfunction and arrhythmias may be poorly tolerated requiring prompt treatment. Negatively inotropic drugs must be used with great care in this group for fear of exacerbating ventricular dysfunction.[29] Trauma to the sinuatrial junction at the time of operation may result in sinus node dysfunction and conversion from a rapid tachycardia may disclose an inadequate recovery response from the sinus node. Children who have undergone the Mustard or Senning procedure for transposition of the great arteries, or those who have survived the Fontan procedure most commonly used for tricuspid atresia or double inlet left ventricle, are frequent sufferers of recurrent atrial arrhythmias, often in the setting of sinus node dysfunction, so called tachybrady syndrome.[30 31] Such children present very difficult management problems and conversion from tachycardia should ideally only be attempted if facilities are available for temporary pacing.

Prognosis

After the diagnosis and successful termination of the first attack the prognosis for children with atrioventricular re-entry tachycardia presenting in the first month of life and early infancy is good and many have no further attacks.[1 2] However, we frequently chose to treat all children for the first year of their lives. After this, they are better able to communicate their problem and are less likely to become haemodynamically compromised should they develop further episodes of supraventricular tachycardia. The cause of the apparent resolution of the condition is unclear. Some authors have suggested that the accessory pathway in such children disappears completely as if the heart were completing its developmental process after birth. Other authors have suggested that the accessory pathway merely becomes dormant and have shown that the triggers required to initiate the re-entry circuit change with increasing age.[32 33]

In terms of freedom from recurrences of arrhythmias the long term prognosis for children presenting later in their lives is not as good as in those infants presenting with the first year as they are less likely to 'grow out' of their tachycardias.[2] Even children with atrioventricular re-entry tachycardia and an otherwise structurally normal heart, presenting at this age, tend to continue having attacks of supraventricular tachycardia; their accessory connection persists. The majority can still be controlled with the commonly available antiarrhythmic agents and in this country drug treatment remains the mainstay of treatment, although we recognise that surgical interruption or ablation of arrhythmogenic substrates such as accessory pathways is successful even in the very young.[34 35] The high success rate and low morbidity now achieved with surgery make this an increasingly appealing option, offering these children a potential cure rather than life long

palliation. Arrhythmias in children with structural heart disease remain a difficult problem. Treatment in this group remains far from ideal and arrhythmias continue to threaten the lives of such children.

An increased interest in arrhythmias in the young and the advent of electrophysiological studies in children has allowed a better understanding of the mechanisms underlying childhood supraventricular tachycardia. This in turn has paved the way for a more logical approach to treatment. With careful attention to the electrocardiogram and a working knowledge of the therapeutic options available, deaths during this hazardous initial period can hopefully be avoided.

Adenosine does not yet have a product license but is available in the United Kingdom. For further information please contact the pharmacy, Royal Brompton and National Heart Hospital.

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